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Application Number Information

Application Number: **09/060659** **Order This File** **Assignments**
Examiner Number: **73462 / PARK, HANKYEL**
Filing Date: **04/15/1998**
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Third Level Review: **NO**
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Status: **150 /PATENTED CASE**
Status Date: **10/20/2000**
Confirmation Number: **9848**
Oral Hearing: **NO**
Title of Invention: **NOVEL EXPRESSION VECTORS AND METHODS OF USE**

Bar Code	PALM Location	Location Date	Charge to Loc	Charge to Name	Employee Name	Location
09060659	9200	03/30/2004	No Charge to Location	No Charge to Name	KASAH,EMMANUEL	

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coprecipitation of sFvTacKDE1 with the 40 kDa precursor

Together, these findings suggest that the absence of mature p55 at the cell surface is due to retention of the immature p40 form in the ER, as a complex with sFvTacKDE1.

These results have been reproduced in two other T leukemic cell lines which express high levels of IL-2Rα. HUT102, an HTLV-1 transformed line, and the Kit225 line. The Kit225 cells are growth factor dependent and were maintained on 100 units/ml IL-7. After introduction of the HIV-1 forced expression vector into these cell lines, a majority of the bulk transduced population were negative for IL-2Rα expression. By subcloning from the bulk population, homogenous negative clones were obtained (FIG. **6**).

Efficient downregulation of IL-2Rα and reduced IL-2 responsiveness in PBMCs transduced with the sFvTacKDEL forced expression vector.

FIG. **7** shows IL-2Rα expression on PHA-activated peripheral blood mononuclear cells transfected with the sFvTacKDEL forced expression vector or a control vector (1) bulk population (FIGS. **7**A–C); or (2) single cell subclones (FIGS. **7**D–G). Flow cytometric analysis of a bulk PBMC population transduced with the HIV-1 forced expression vector showed virtually no IL-2Rα expression (FIG. **7**B) in comparison to a control population transduced with an irrelevant (empty) vector HVSL3P (FIG. **7**C). These cells were generated and maintained in the presence of IL-7, an alternative T cell growth factor. By subcloning (FIG. **7**B), single cell clones were obtained that express no detectable IL-2Rα (See FIGS. **7**E and F). A thymidine incorporation assay was used to measure the IL-2 responsiveness of the IL-2Rα negative clones (FIG. **8**). FIG. **8** shows IL-2 induced proliferation in peripheral blood T cell clones which are positive (clone 5) or negative (clone 2) for IL-2Rα expression. These clones did not respond to low doses of IL-2 (1 unit/ml). Some proliferation was seen at doses of 10 and 100 units/ml but when compared to an IL-2Rα-positive clone, ~10 times more IL-2 was required to achieve an equivalent proliferative response. Some IL-2 responsiveness was expected, even in the absence of IL-2Rα, as these cells will still express intermediate affinity receptors for IL-2. These data demonstrate functional as well as phenotypic evidence for the absence of high affinity IL-2 receptors in the IL-2Rα negative cells.

We claim:

1. A lentiviral vector containing a gene of interest operably linked to a selectable marker gene by an internal ribosome entry site (IRES).

2. The lentiviral vector of claim **1**, wherein the gene of interest is a gene whose expression in a mammalian cell is selected against as determined by comparing a cell transduced using a divalent vector or co-transfection with a selectable marker and said gene, with a control cell transduced using a divalent vector or co-transfection with only said selectable marker.

3. The lentiviral vector of claim **2**, wherein the gene of interest is selected from the group consisting of a gene for HTLV-1 tax, HTLV-2 tax, an antibody and a protein that is part of a multi-tiered expression system.

4. The lentiviral vector of claim **1**, wherein a defective lentiviral vector is used.

5. The lentiviral vector of claim **4**, wherein the defective lentiviral vector is a lentiviral vector containing multiple splice donor and splice acceptor sites.

6. The lentiviral vector of claim **5**, wherein the lentiviral vector is an HIV viral vector.

7. A method of using the vector of claim **1** to obtain forced expression of the gene of interest which comprises using the vector of claim **1** to transduce a mammalian cell, culturing the transduced cell under conditions sufficed to express the selectable marker gene, and then exerting selection pressure on the transduced cell to select for that selectable marker.

8. The lentiviral vector of claim **3**, wherein the gene of interest is an antibody gene.

9. The lentiviral vector containing two different genes linked together by an internal ribosome entry site (IRES).

* * * * *